REVIEW ARTICLE

Screening for Sleep Apnea in Posttraumatic Stress Disorder

R. Gregory Lande, DO; Cynthia Gragnani, Ph.D
Walter Reed National Military Medical Center, Bethesda, MD

KEYWORDS:
PTSD
Obstructive Sleep Apnea
Home Sleep Study
Military
Psychiatry

Sleep problems are one of the most enduring complaints from individuals with post-traumatic stress disorder. In this study, the investigators explored the relationships between a commonly administered post-traumatic stress disorder screening instrument, the Posttraumatic Stress Disorder Checklist – Military Version (PCLm) and results obtained from home sleep studies obtained from active duty service members. This retrospective study was conducted among active duty service members receiving care on the Psychiatry Continuity Service (PCS) at Walter Reed National Military Medical Center. The investigators examined 135 records of subjects referred for an enhanced sleep assessment from October 1, 2010 through November 30, 2013. There were significant direct correlations between the PCLm score and the sleep assessment values: wake percent (n=121, p=.022).onset of first deep sleep (n=106, p=.024) the apnea/hypopnea index (n=110, p=.028) and the oxygen desaturation index (n=110, p=.025).

INTRODUCTION

The diagnostic criteria for posttraumatic stress disorder (PTSD) involve a pathologic constellation following a traumatic event that involves intrusive recollections, avoidance of triggering stimuli, negative thinking, and hyperarousal.[1] Some of the more troubling complaints cluster around the person's sleep, such as intrusive nightmares, problems falling asleep due to hyper vigilance, and even futile efforts to avoid sleep. The inextricable link between PTSD and sleep problems has led some researchers to speculate that PTSD is fundamentally a sleep disorder.[2] As researchers continue to probe the relationship between PTSD and sleep, other evidence is emerging suggesting that abnormalities in the sleep cycle, specifically rapid eye movement (REM) sleep may be associated with the persistence of troubling dreams and disruptive behaviors while abnormalities in non-rapid eye movement (NREM) are more likely related to insomnia.[3,4]

One of the frustrations for clinicians treating PTSD is the intractability of the disorder. Since sleep is such a central component of PTSD it would seem reasonable to include a detailed sleep assessment in each case to ensure treatment planning incorporated this element. Doing so may help improve one of the most common complaints of PTSD patients. The value of such an approach is increasingly suggested, by emphasizing for example, initial medication management that includes trazodone and prazosin that more selectively target sleep fragmentation and nightmares. [5,6] Sleep problems such as insomnia and nightmares also independently contribute to a heightened risk of suicide, which when added to PTSD, offers an even more potent argument for addressing this issue.[7]

A more obscure association with PTSD that is getting more research attention is the apparent increased incidence of obstructive sleep apnea (OSA). This relationship has come to the attention of researchers studying service members with combat related PTSD suggesting that the incidence and severity of their PTSD may be related to OSA.[8-10] Taking a deep dive into the specific parameters affected through this relationship reveals changes in the sleep cycle of combat veterans, such as a significant reduction in both REM and NREM and more severe breathing problems than seen in non combat related PTSD cases.

In another study, the authors investigated sleep complaints among service members with combat related PTSD and through the use of polysomnography determined that two-thirds of the subjects met the diagnostic criteria for OSA.[11] Interestingly, the same study reported that OSA was significantly higher among service members without accompanying physical injuries, leading to speculation that an undiagnosed, pre-existing breathing problem might be a risk factor for the subsequent development of PTSD.

Similar findings have been reported among individuals with PTSD from other traumas such as crime, natural disasters, and terrorism.[12-15] In these cases, the OSA-PTSD dyad differs from non trauma related breathing problems with PTSD patients more commonly complaining of nightmares, difficulty initiating sleep, and an overall less satisfying night's sleep.

Despite newer studies increasingly pointing to a higher rate of OSA among individuals with PTSD, the exact etiology possibly uniting the pair in not yet well understood. Even so, it seems clear that recognizing and simultaneously treating both conditions will improve combat related PTSD[16,17] More specifically, one of the most distressing symptoms arising from PTSD involves the endless repetition of nightmares, a condition that may improve when the OSA is appropriately managed.[17-20]

In this study, the investigators explored the relationships between a commonly administered PTSD screening instrument and results obtained from home sleep studies obtained from active duty service members.

METHODS

This retrospective study was conducted among active duty service members receiving care on the Psychiatry Continuity Service (PCS) at Walter Reed National Military Medical (WRNMMC) Center. The PCS is a partial hospital program providing eight hours of clinical activities five days a week with an average length of stay of approximately one month, adjusted as necessary by the treatment plan. As a tertiary care facility WRNMMC tends to receive more complicated cases, a trend mirrored on the PCS. The most common diagnoses include all depressive disorders, PTSD, and substance use disorders. Sleep problems are one of the most common concerns, leading to the implementation of an enhanced sleep assessment that includes a home sleep study.

Home sleep devices allow patients to do sleep studies at home. These portable home sleep devices are sophisticated instruments capable of measuring several physiologic parameters that produce objective information about a person's sleep. The commercially available devices vary in their capacity to report different factors such as accurate respiratory data, the sleep-wake cycle, and the actual sleep time. The better home sleep monitors rely in part on the well-established relationship between fluctuations in the blood pressure and changes in the sleep cycle.[21] The sleep cycle is characterized by predictable changes in blood pressure, most dramatically represented by the rise in blood pressure that accompanies REM sleep.[22] Cardiac activity changes through the phases of the sleep cycle.[23] For example, during REM sleep the heart rate and blood pressure all increase. The connection between autonomic activity and the phases of the sleep-wake cycle awaited technological innovations that could reliably measure these subtle physiologic changes. One important advance was the development of a finger mounted plethysmographic sensor to measure peripheral arterial tone (PAT). The PAT sensor is constructed to monitor the decrease venous engorgement while simultaneously unloading arterial wall tension, thereby promoting the dynamic range of the device.[24]

Study investigators used the WatchPAT™, a commercially available FDA approved medical device (Itamar Medical, Caesarea, Israel) for the home sleep studies. This medical device is a wrist-worn device that captures information from the PAT sensor, an actigraph, and a finger mounted pulse oximeter; and then stores the data on a secure digital card through the duration of the sleep study.

In a study comparing the WatchPat™ with concurrently administered polysomnography (PSG), the authors' reported a significant correlation (r=0.87, P<0.001) between the two procedures in detecting arousals.[25] The same device was the subject of another study examining the accuracy of the WatchPAT™ in diagnosing obstructive sleep apnea (OSA). The researchers reported a significant agreement (r=0.87, P<0.001) between the results obtained through PSG and the WatchPAT™.[26] In another study, researchers assessing the accuracy of respiratory parameters produced by this device reported that it was, "accurate, robust, and reliable ambulatory method for the detection of…" obstructive sleep apnea.[27]

Researchers have tested the actigraph portion of the WatchPAT™ and reported reasonable accuracy, versus PSG, in measuring wakefulness and sleep.[28] The device was capable of identifying respiratory values used by Medicare for diagnosing OSA.[29] In a study comparing the apnea-hypopnea index (AHI) as reported from a PSG versus a simultaneous recording from the WatchPat, the authors reported a significant (r=.90, P< .0001) agreement.[30] Another study comparing PSG with the WatchPAT™ also reported significant (r=.93, P< .0001) agreement on the AHI.[31]

Reasonable clinical guidelines for the use of portable home sleep monitors emphasize the need for a comprehensive clinical assessment, particularly for the common co-occurring problems in the study group. In cases where the monitors are used exclusively to diagnose OSA, the guidelines recommend close collaboration with sleep medicine experts.[32]

In this study, the investigators used the WatchPAT 200™ to conduct the home sleep studies. This FDA approved medical device calculates the severity of sleep apnea through three measurements, the apnea/hypopnea index (AHI), oxygen desaturation index (ODI), and the respiratory disturbance index (RDI). The AHI represents the total number of complete cessations (apneas) and partial obstructions (hypopneas) of breathing per hour of sleep. An AHI score from 5-14 indicates mild OSA, 15-30 moderate and severe is greater than 30.[33] The ODI measures changes in blood oxygenation from baseline. The RDI assesses the severity of sleep apnea by measuring respiratory efforts, or RERAs (Respiratory Effort Related Arousals). A RERA is an arousal from sleep that follows 10 seconds or more of increased respiratory effort

Address correspondence to: R. Gregory Lande, DO*, Psychiatry Continuity Service, Walter Reed National Military Medical Center. 8901 Rockville Pike, Bethesda, MD 20889-5600; Phone: (301) 400-2110; Email: rglande@gmail.com

1877-5773X/$ - see front matter. © 2015 ACOFP. All rights reserved.

that does not meet the criteria for apnea or hypopnea.[34]

The investigators compared the home sleep study results with the Posttraumatic Stress Disorder Checklist – Military Version, (PCLm).[35] The PCLm is a 17-item self-report instrument from which subjects choose among 5 descriptions:

1= Not at all

2= A little bit

3= Moderately

4 - Quite a bit

5 = Extremely

A typical question from the PCLm asks the service member if they are "Having physical reactions when something reminded you of a stressful military experience from the past." For purposes of this study, scores above 49 suggest that the symptoms are consistent with the clinical diagnosis PTSD.

PCS patients were scheduled for an enhanced sleep assessment based on the results of the Pittsburg Insomnia Rating Scale (PIRS). The PIRS is a 20-item self-report instrument assessing sleep over the preceding 7-day period.[36] The range of scores on the PIRS is from 0-60 with scores above 20 suggesting insomnia. Typical questions on the PIRS include: "From the time you tried to go to sleep, how long did it take to fall asleep on most nights?" and "If you woke up during the night, how long did it take to fall back to sleep on most nights?"

In addition to the PIRS, all patients referred for a more detailed sleep assessment had their basal metabolic index (BMI) calculated and completed the Alcohol Use Disorders Identification Test (AUDIT)[37] The AUDIT consists of ten questions and five responses per item. Typical questions include, "How often do you have a drink containing alcohol?" and "How often do you have six or more drinks on one occasion?" In responding to these questions, subjects could choose from "never "which scored 0 for that scale item, "monthly or less (1)", "2-4 times a month (2)", "2-3 times a week (3) ", and "4 or more times a week (4)" which earned the maximum score for that scale question of four. Scores exceeding seven are associated with harmful drinking.

Data were analyzed using Statistical Package for the Social Sciences (SPSS) version 20.

RESULTS

The investigators examined 135 records of subjects referred for an enhanced sleep assessment from October 1, 2010 through November 30, 2013. Almost two-thirds of the participants

Table 1 - Sleep Results for Total Study Group

Test Name	n*	Mean	SD
PIRS	135	41.10	10.52
AUDIT	111	4.68	7.66
PCLm	122	51.36	18.13
BMI	133	27.10	4.28
REM%	119	20.63	8.35
Deep Sleep%	119	20.81	6.09
Light Sleep%	119	58.56	11.14
Wake%	134	21.25	12.69
Time to Sleep(minutes)	118	25.28	15.60
Time First Deep Sleep(minutes)	118	65.27	48.63
Time First REM Sleep(minutes)	117	150.99	77.38
Sleep Time(minutes)	134	366.68	93.61
Apnea/Hypopnea Index	123	3.78	4.60
Oxygen desaturation index	123	1.66	2.69

*n varies due to missing data

Table 2 - Relationships between PCLm and Sleep Factors

Test		PCLm
AUDIT	Pearson Correlation	.101
	Sig. (2-tailed)	.295
	n*	110
PIRS	Pearson Correlation	.350
	Sig. (2-tailed)	.000***
	n	122
BMI	Pearson Correlation	.053
	Sig. (2-tailed)	.565
	n	120
Time to Sleep	Pearson Correlation	-.006
	Sig. (2-tailed)	.950
	n	106
Sleep Time	Pearson Correlation	-.279
	Sig. (2-tailed)	.002***
	n	121
Wake %	Pearson Correlation	.208
	Sig. (2-tailed)	.022**
	n	121
First Deep	Pearson Correlation	.219
	Sig. (2-tailed)	.024**
	n	106
First REM	Pearson Correlation	.168
	Sig. (2-tailed)	.087
	n	105
Light Sleep %	Pearson Correlation	.116
	Sig. (2-tailed)	.234
	n	107
REM %	Pearson Correlation	-.203
	Sig. (2-tailed)	.036**
	n	107
Deep Sleep%	Pearson Correlation	.068
	Sig. (2-tailed)	.487
	n	107
AHI	Pearson Correlation	209
	Sig. (2-tailed)	.028**
	n	110
ODI	Pearson Correlation	.214
	Sig. (2-tailed)	.025**
	n	110

* n varies based on missing data

**Correlation is significant at the 0.05 level

were male (n=87/135, 64%) and roughly the same percentage of subjects' (n=90/129, 69.8%) age range was between 21-35.

All subjects referred for an enhanced sleep study had evidence of insomnia based on the average PIRS score (n=135, Mean 41.10, SD 10.52), as well as trauma symptoms based on the PCLm (n=122, Mean 51.36, SD 18.13), and a slightly elevated BMI (n=133, Mean 27.10, SD 4.28). Subjects needed almost a half an hour to fall asleep (n=118, Mean 25.28, SD 15.60), had about six hours total time asleep, (n=118, Mean 6.12, SD 1.57) and based on the average AHI (n=123, Mean 3.78, SD 4.60) did not manifest breathing problems while asleep ***(See Table 1)***.

Correlations between the PCLm and various sleep factors revealed several significant findings. Not surprisingly the higher the PCLm score the higher was the PIRS. (n=122, p=.002). Other results included an inverse relationship between total time slept (n=121, p=.002) and the percent of REM sleep (n=107, p=.036). There were significant direct correlations between the PCLm score and the wake percent (n=121, p=.022).onset of first deep sleep (n=106, p=.024). AHI (n=110, p=.028) and the ODI (n=110, p=.025). There were no significant correlations between the PCLm and the AUDIT score or the BMI. ***(See Table 2)***

DISCUSSION

This study confirms what patients bitterly complain about, that sleep problems are inextricably intertwined with PTSD. The main casualties are seen in the inverse relationship between PTSD and total sleep time, as the former goes up the latter goes down. In a similar fashion, the amount of REM sleep also decreases as the symptoms of PTSD increase. The first episode of deep sleep is delayed and in a nearly significant trend the first episode of REM sleep is also pushed later into the sleep cycle as the PCLm scores increase.

Perhaps one of the more interesting correlations is the relationship between PTSD and breathing problems while asleep. Two common parameters of OSA, the AHI and the ODI both increased along with the intensity of PTSD. Undiagnosed OSA could be an important factor complicating PTSD improvement.

Another interesting finding showed no significant relationship between the PClm and the person's BMI or alcohol use as screened by the AUDIT. Both of these findings would benefit from further study since alcohol use and obesity can independently and adversely affect sleep but in this study when compared with the PCLm there were no significant correlations.

Based on the findings in this study clinicians should routinely incorporate screening tests to assess trauma symptoms and sleep. Not every patient with PTSD will need a PSG or a home sleep study but as revealed in this study the higher the PCLm score the more likely is the possibility that a significant problem, such as OSA is present.

There are limitations in this study. The investigators used the PCLm that is tailored for military trauma. Other versions of the instrument are available, and while the investigators believe they would result in similar findings, this opinion would benefit from research. Also, the subjects in this study had combat-related PTSD, a stressor that may be specific enough to affect the results. Other research might address different trauma types.

CONCLUSION

For the typical patient with PTSD, sleep problems often lead the list of enduring complaints. A poor night's sleep dominated by frequent awakenings, troubling dreams, and short duration may suggest more significant underlying pathology such as OSA. A simple screening tool used in this study, the WatchPat™ could help clinicians more accurately predict the problems, tailor therapy, or seek referral as needed.

The views expressed in this article are those of the authors and do not reflect the official policy of the Department of Army/Navy/Air Force, Department of Defense. The authors are not endorsing any commercial product.

References

1. Association AP. Diagnostic and statistical manual of mental disorders (5th ed.). Arlington, VA: American Psychiatric Publishing; 2013.
2. Morrison AR. Sleep disturbance as the hallmark of posttraumatic stress disorder. Am J Psychiatry. 1989;146(6):697-707.
3. Harvey AG, Jones C, Schmidt DA. Sleep and posttraumatic stress disorder: a review. Clinical Psychology Review. 2003;23(3):377-407.
4. Talbot L, Neylan T, Metzler T, Cohen B. The Mediating Effect of Sleep Quality on the Relationship between PTSD and Physical Activity. Journal of clinical sleep medicine: JCSM: official publication of the American Academy of Sleep Medicine. 2013;10(7):795-801.
5. Bajor LA, Ticlea AN, Osser DN. The Psychopharmacology Algorithm Project at the Harvard South Shore Program: an update on posttraumatic stress disorder. Harvard review of psychiatry. Sep-Oct 2011;19(5):240-258.
6. Raskind MA, Peterson K, Williams T, et al. A trial of prazosin for combat trauma PTSD with nightmares in active-duty soldiers returned from Iraq and Afghanistan. Am J Psychiatry. Sep 1 2013;170(9):1003-1010.
7. Lande RG, Gragnani C. Sleep trends of active-duty service members referred for psychiatric care: a descriptive study. The Journal of the American Osteopathic Association. Feb 2013;113(2):144-150.
8. Mysliwiec V, Matsangas P, Baxter T, McGraw L, Bothwell NE, Roth BJ. Comorbid insomnia and obstructive sleep apnea in military personnel: correlation with polysomnographic variables. Military medicine. 2014;179(3):294-300.
9. Capaldi VF, Guerrero ML, Killgore WD. Sleep disruptions among returning combat veterans from Iraq and Afghanistan. Military medicine. 2011;176(8):879-888.

10. Lande RG. Sleep Problems, Posttraumatic Stress, and Mood Disorders Among Active-Duty Service Members. JAOA: Journal of the American Osteopathic Association. 2014;114(2):83-89.
11. Williams SG, Collen J, Orr N, Holley AB, Lettieri CJ. Sleep disorders in combat-related PTSD. Sleep and Breathing. 2014:1-8.
12. Krakow B, Melendrez D, Warner TD, et al. Signs and symptoms of sleep-disordered breathing in trauma survivors: a matched comparison with classic sleep apnea patients. The Journal of nervous and mental disease. Jun 2006;194(6):433-439.
13. Krakow B, Haynes PL, Warner TD, et al. Nightmares, insomnia, and sleep-disordered breathing in fire evacuees seeking treatment for posttraumatic sleep disturbance. Journal of traumatic stress. Jun 2004;17(3):257-268.
14. Webber MP, Lee R, Soo J, et al. Prevalence and incidence of high risk for obstructive sleep apnea in World Trade Center-exposed rescue/recovery workers. Sleep and Breathing. 2011;15(3):283-294.
15. Sharafkhaneh A, Giray N, Richardson P, Young T, Hirshkowitz M. Association of psychiatric disorders and sleep apnea in a large cohort. SLEEP-NEW YORK THEN WESTCHESTER-. 2005;28(11):1405.
16. Krakow B, Ulibarri VA, Moore B, McIver ND. Posttraumatic Stress Disorder and Sleep-Disordered Breathing: A Review of Comorbidity Research. Sleep Medicine Reviews. 2014.
17. Gupta M, Simpson F. Obstructive Sleep Apnea and Psychiatric Disorders: A Systematic Review. Journal of clinical sleep medicine: JCSM: official publication of the American Academy of Sleep Medicine. 2014.
18. Tamanna S, Parker J, Lyons J, Ullah M. The Effect of Continuous Positive Air Pressure (CPAP) on Nightmares in Patients with Posttraumatic Stress Disorder (PTSD) and Obstructive Sleep Apnea (OSA). Journal of clinical sleep medicine: JCSM: official publication of the American Academy of Sleep Medicine. 2013;10(6):631-636.
19. El-Solh AA, Ayyar L, Akinnusi M, Relia S, Akinnusi O. Positive airway pressure adherence in veterans with posttraumatic stress disorder. Sleep. Nov 2010;33(11):1495-1500.
20. BaHammam AS, Al-Shimemeri SA, Salama RI, Sharif MM. Clinical and polysomnographic characteristics and response to continuous positive airway pressure therapy in obstructive sleep apnea patients with nightmares. Sleep medicine. 2013;14(2):149-154.
21. Littler WA, Honour AJ, Carter RD, Sleight P. Sleep and blood pressure. Br Med J. Aug 9 1975;3(5979):346-348.
22. Somers VK, Dyken ME, Mark AL, Abboud FM. Sympathetic-nerve activity during sleep in normal subjects. N Engl J Med. Feb 4 1993;328(5):303-307.
23. Trinder J, Kleiman J, Carrington M, et al. Autonomic activity during human sleep as a function of time and sleep stage. J Sleep Res. Dec 2001;10(4):253-264.
24. Lavie P, Schnall RP, Sheffy J, Shlitner A. Peripheral vasoconstriction during REM sleep detected by a new plethysmographic method. Nat Med. Jun 2000;6(6):606.
25. Pillar G, Bar A, Betito M, et al. An automatic ambulatory device for detection of AASM defined arousals from sleep: the WP100. Sleep Med. May 2003;4(3):207-212.
26. Ayas NT, Pittman S, MacDonald M, White DP. Assessment of a wrist-worn device in the detection of obstructive sleep apnea. Sleep Med. Sep 2003;4(5):435-442.
27. Bar A, Pillar G, Dvir I, Sheffy J, Schnall RP, Lavie P. Evaluation of a portable device based on peripheral arterial tone for unattended home sleep studies. Chest. Mar 2003;123(3):695-703.
28. Hedner J, Pillar G, Pittman SD, Zou D, Grote L, White DP. A novel adaptive wrist actigraphy algorithm for sleep-wake assessment in sleep apnea patients. Sleep. Dec 15 2004;27(8):1560-1566.
29. Pittman SD, Ayas NT, MacDonald MM, Malhotra A, Fogel RB, White DP. Using a wrist-worn device based on peripheral arterial tonometry to diagnose obstructive sleep apnea: in-laboratory and ambulatory validation. Sleep. Aug 1 2004;27(5):923-933.
30. Zou D, Grote L, Peker Y, Lindblad U, Hedner J. Validation a portable monitoring device for sleep apnea diagnosis in a population based cohort using synchronized home polysomnography. Sleep. Mar 2006;29(3):367-374.
31. Pang KP, Gourin CG, Terris DJ. A comparison of polysomnography and the WatchPAT in the diagnosis of obstructive sleep apnea. Otolaryngology--head and neck surgery : official journal of American Academy of Otolaryngology-Head and Neck Surgery. Oct 2007;137(4):665-668.
32. Collop NA, Anderson WM, Boehlecke B, et al. Clinical guidelines for the use of unattended portable monitors in the diagnosis of obstructive sleep apnea in adult patients. Portable Monitoring Task Force of the American Academy of Sleep Medicine. J Clin Sleep Med. Dec 15 2007;3(7):737-747.
33. Patton LL, Association AD. The ADA Practical Guide to Patients with Medical Conditions. Wiley; 2012.
34. The Key to Treatment is Proper Diagnosis. http://www.itamar-medical.com/WatchPAT/Patient/WatchPAT/Complete_Overview.html. Accessed January 21, 2013.
35. Blanchard EB, Jones-Alexander J, Buckley TC, Forneris CA. Psychometric properties of the PTSD Checklist (PCL). Behav Res Ther. Aug 1996;34(8):669-673.
36. Moul DE, Pilkonis,P.A., Miewald,J.M., Carey,T.J., Buysse,D.J. Preliminary study of the test-retest reliability and concurrent validities of the Pittsburgh Insomnia Rating Scale (PIRS). Sleep. 2002;25(Abstract Supplement,):246-247.
37. Saunders JB, Aasland OG, Babor TF, de la Fuente JR, Grant M. Development of the Alcohol Use Disorders Identification Test (AUDIT): WHO Collaborative Project on Early Detection of Persons with Harmful Alcohol Consumption--II. Addiction. Jun 1993;88(6):791-804.

Osteopathic Family Physician (2015) 39-42

REVIEW ARTICLE

Wound Tetanus

William Woolery DO PhD MS FACOFP
Hospitalist Director, Sacred Heart Hospital on the Gulf, Port St. Joe, FL.

KEYWORDS:
Tetanus
Antibiotic Therapy
HTIG

We report a case of wound tetanus in a previously immunized patient. The patient developed generalized tetanus requiring IV antibiotic therapy & human tetanus immune globulin (HTIG) therapy. This is only the 15th case reported this year in the United States.

INTRODUCTION

Tetanus occurs worldwide. It is a common problem in areas of the world that are densely populated, and in hot climates in which the soil is rich in organic matter. Reported cases occur more frequently in underdeveloped, overcrowded and economically disadvantaged countries. The disease has been described in the Bible and ancient writings of Greek and Egyptian physicians. It is the only vaccine preventable disease that is infectious but not contagious.

Approximately seventy-five percent of cases occur between April and October. There are between 800,000 and one million cases worldwide yearly. Worldwide deaths have been reported between 210,000 to one million yearly. Greater than fifty percent of these deaths are from neonatal tetanus. Many cases go unreported each year.[1,2,3,9]

In 1903 there were 406 deaths reported from tetanus due to infections obtained from 3983 hand injuries on the fourth of July from fireworks. This led the American Medical Association to recommend banning hand held fireworks. Prophylaxis against tetanus began in World War I. Immunization programs for the military were in place by 1924 and were routine by 1946. All United States military dog tags from 1940 bore the date of the soldier's tetanus immunization. There were no reported military cases of tetanus during the Vietnam War.

Since 2000 there has been an average of approximately thirty cases per year in the United States. The mortality rate has decreased from ninety-one percent in 1947 to 13-42% today. In the early-to-mid 1940's nationwide immunization programs were instituted in the United States. Tetanus became a reportable disease in 1947. The 560 cases reported yearly have dramatically decreased directly due to the institution of immunization protocols. Only 14 cases were reported in 2014. There have been zero deaths in those patients who have completed a primary immunization series.[4,7]

Risk factors for the development of tetanus include: age > 60, short incubation period, inadequate tetanus toxoid vaccination, tetanus prone wounds, intravenous drug use (IVDU), diabetes mellitus, chronic venous stasis ulcers. Currently most cases if tetanus in the United States occur in patients with a history of under immunization. At greater risk are heroin IVDU and older adults because of their higher rate of being unvaccinated or under vaccinated.

PATHOGENESIS

Tetanus is caused by a gram-positive obligate anaerobic spore forming bacillus, Clostridium tetani. Spores of C. tetani are ubiquitous in nature. They have been found in the gastrointestinal tract of humans and domesticated animals, soil, house dust, fresh and salt water. The spores are highly resistant to temperature extremes and humidity and can survive indefinitely. The spores will not germinate unless adequate anaerobic conditions are present. When favorable tissue conditions exist the spores germinate to form mature bacilli which produce exotoxins tetanolysin and tetanospasmin.

Tetanolysin has an undefined role in the development of clinical tetanus. It is thought to contribute to the development of localized anaerobic tissue conditions by direct damaging effects on traumatized tissue. However, the exact mechanism by which this process takes place is still undetermined.[5,6]

Tetanospasmin is second only to botulinum toxin in potency and is responsible for the clinical manifestation(s) of the disease. Tetanus toxoid is an inactivated form of tetanospasmin. The majority of toxin production occurs at the end of the germination phase which only occurs under strict anaerobic conditions. This exotoxin enters peripheral nerves and via the axonal retrograde transport system is transported

Address correspondence to: William Woolery, DO, Sacred Heart Hospital on the Gulf - Hospitalist, 3801 E. Highway 98, Port St. Joe, FL 32456. Email: william.woolery@shhpens.org

1877-5773X/$ - see front matter. © 2015 ACOFP. All rights reserved.